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Right Coronary Spasm Complicating Electrode Catheter Ablation of a Right Lateral Accessory Pathway

GEOFFREY O. HARTZLER, MD, FACC, LEE V. GIORGI, MD, ANTONI M. DIEHL, MD, FACC, WILLIAM R. HAMAKER, MD, FACC

Kansas City, Missouri

Severe focal right coronary artery spasm, demonstrated on angiography, occurred in a 12 year old girl undergoing attempted electrode catheter ablation of a right atrial-right ventricular free wall accessory pathway.

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Electrode catheter ablation of the atrioventricular (AV) conduction system, accessory pathways, arrhythmogenic foci and localized reentrant circuits has been described (1–5). However, experience with this technique is limited and potential complications remain to be elucidated. We report the occurrence of angiographically severe coronary artery spasm after attempted electrode catheter ablation of an accessory pathway.

Case Description

In July 1984, a 12 year old girl underwent attempted electrode catheter ablation of a right atrial-right ventricular free wall accessory pathway. Two years previously, because of recurring episodes of a wide complex tachycardia associated with dizziness and near syncope, she underwent invasive electrophysiologic study. Although the rest electrocardiogram was normal, anterograde preexcitation was documented during incremental atrial pacing and programmed atrial stimulation and during wide complex supraventricular tachycardia. The presence of a lateral right atrial-right ventricular accessory pathway was confirmed by extensive endocavitary mapping. Retrograde conduction by the accessory pathway was absent. Short-term testing with class I antiarrhythmic drugs resulted in prolonged refractoriness within the accessory pathway and a markedly increased tachycardia cycle length. Subsequently, she received multiple oral antiarrhythmic drugs including quinidine, disopyramide, propranolol and digitalis, but continued to experience episodes of symptomatic tachycardia and drug-induced side effects.

Ablation procedure. After parental informed consent was obtained, the patient underwent attempted electrode catheter ablation. With general endotracheal anesthesia, baseline coronary arteriography demonstrated a normal and dominant right coronary artery (Fig. 1A). Repeat electrophysiologic study confirmed previous observations. The tip electrode of a 7F quadripolar electrode catheter (USCI) was positioned at the AV groove immediately adjacent to the site of earliest right ventricular activation during atrial pacing and supraventricular tachycardia (Fig. 1B). Using a standard defibrillator (Lifepak 6), an initial 50 J discharge was delivered from the tip electrode directed to a right anterolateral chest paddle. Repeat atrial pacing demonstrated the persistence of pre-excitation. A second 200 J discharge was delivered, resulting in loss of ventricular preexcitation during atrial pacing and programmed atrial stimulation, preventing the reinduction of supraventricular tachycardia. Two additional 200 J discharges were delivered to the same location. After each discharge, there was marked but transient ST segment elevation in all electrocardiographic monitor leads (V_1, I and aVF), returning to baseline within 3 to 5 minutes. After all shocks, repeat atrial pacing demonstrated complete block within the accessory pathway.

Fifteen minutes after the final intracardiac shock, repeat right coronary arteriography showed subtotal occlusion of the posterior descending branch and high grade narrowing of the posterolateral branch of the right coronary artery (Fig. 2A). Intracoronary nitroglycerin, 400 μg, was administered in two boluses and repeat right coronary injections demonstrated decreased localized obstruction (Fig. 2B).

Post-procedure observations and management. After the procedure, the patient was maintained on continuous

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Address for reprints: Geoffrey O. Hartzler, MD, Medical Plaza 11-20, 4320 Wornall Road, Kansas City, Missouri 64111.

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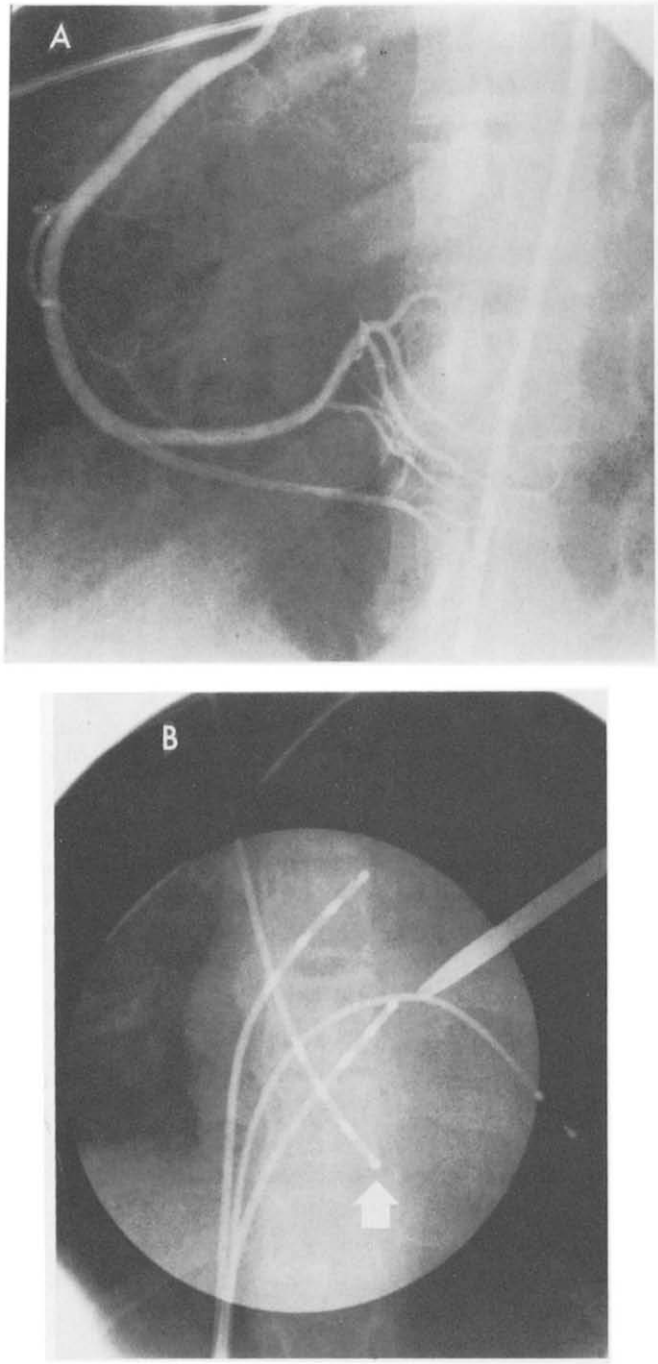


Figure 1. A, Baseline right coronary arteriogram demonstrating dominant circulation with a small posterior descending branch and a large posterolateral branch. **B**, Catheter positions during the ablation procedure. The tip (**arrow**) of a 7F quadripolar electrode catheter is positioned immediately adjacent to the posterolateral right AV groove at the site of earliest endocardial activation. All shocks were delivered from the tip electrode of this catheter directed to a chest paddle which is also visualized in this X-ray film.

intravenous heparin, transcutaneous nitrates and an oral calcium channel blocking agent. However, 6 hours later, spontaneous wide complex supraventricular tachycardia occurred, confirming the return of anterograde pre-excitation. Serum creatine kinase (CK) reached 2,290 mlU/ml (normal 50 to 150) with MB fraction present, but not quantitated. However, a pyrophosphate scan of the heart obtained at 48 hours showed no localized uptake, and all 12 lead electrocardiograms remained normal and unchanged.

Forty-eight hours after the procedure, a limited electrophysiologic study was performed, again confirming the return of anterograde pre-excitation. Left ventriculography demonstrated generalized dynamic contractility, but mild hypokinesia of the mid and apical diaphragmatic segments. Right coronary arteriography showed wide patency with no focal abnormalities of the distal vessel or branches (Fig. 2C).

Surgery. Seventy-two hours after the ablation procedure, the patient underwent successful operative interruption of a right lateral free wall accessory pathway. Intraoperative inspection revealed no gross abnormalities attributable to the previous catheterization procedure. Intraoperative epicardial mapping suggested the presence of a relatively broad band of accessory conduction tissue. The postoperative course was uneventful, with late electrophysiologic study confirming the absence of preexcitation.

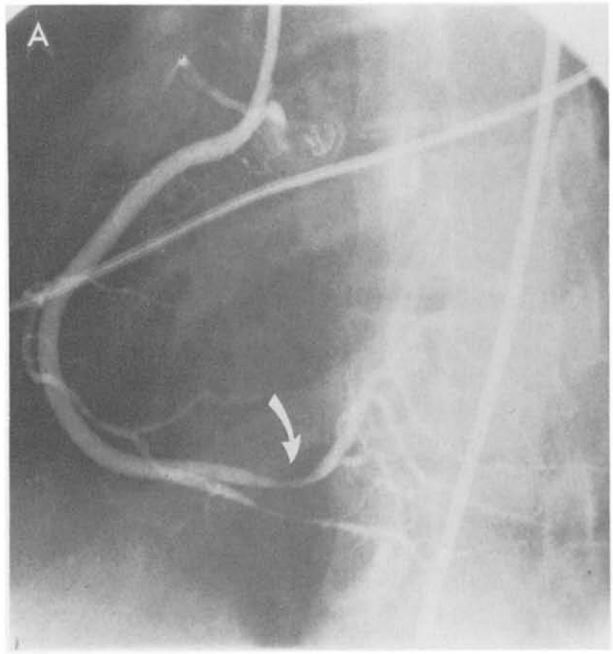
Discussion

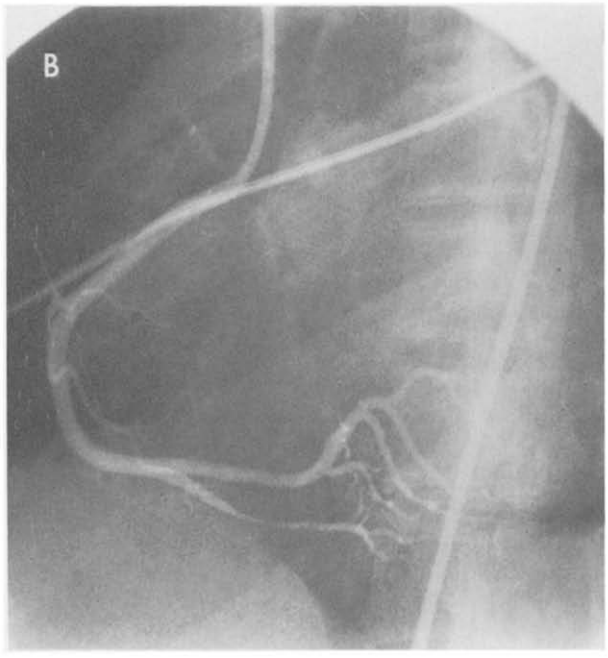
Previous ablation reports. Morady and Scheinman (4) described successful long-term electrode catheter ablation of an accessory pathway located within the posterior interatrial septum. Successful interruption of a free wall right atrial-right ventricular or left atrial-left ventricular accessory pathway has been attempted but not accomplished (6–8).

Brodman and Fisher (9) described the pathologic sequelae of relatively low energy (35 to 45 J) and higher energy (240 J) discharges within the canine coronary sinus. In this model, observations included the occurrence of acute coronary sinus rupture, extensive ecchymosis and edema surrounding the coronary sinus extending into adjacent left atrial and ventricular walls, late dense scarring of the left atrial wall adjacent to shock sites and mild stenosis to complete occlusion of the coronary sinus at sites of injury. Additionally, three dogs receiving 240 J discharges had mild to marked intimal hyperplasia within the immediately adjacent circumflex coronary artery.

Myocardial damage and coronary spasm. In our experience with ablation of the AV conduction system and arrhythmogenic sites within the ventricles, repeated discharges have proved more effective than a single electrical discharge. Because of the relative imprecision of electrode catheter mapping and because of the intramyocardial and alleged occasional epicardial location of accessory fibers, one would expect that higher energy and repeated electrical shocks would be required to permanently ablate an accessory pathway.

We have found that repeated high energy intracardiac





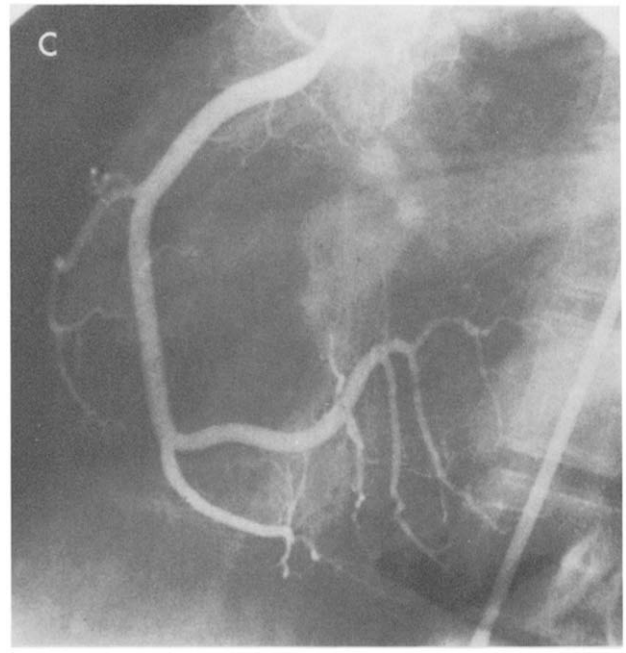


Figure 2. Post-ablation right coronary arteriograms. **A**, Immediately after the procedure, showing diffuse subtotal narrowing of the posterior descending branch and high grade obstruction of the posterolateral branch (**arrow**). **B**, After 400 μg of intracoronary nitroglycerin, lessening of spasm is evident in both branches even though the posterior descending branch itself remains tightly narrowed. **C**, Forty-eight hours after the ablation procedure in the absence of nitroglycerin, calcium channel blocking agents or vasodilators. The sites of previous focal spasm are now angiographically normal.

shocks typically result in total CK values ranging 1,000 to several thousand mIU/ml. As with repeated transthoracic direct current countershocks, the vast majority of the CK is of skeletal muscle origin, although the MB fraction, when quantitated, may reach 10 to 12%, reflecting the anticipated myocardial damage or "infarction." The CK values and absence of 12 lead electrocardiographic changes in our patients are consistent with an uncomplicated ablation procedure and do not allow the conclusion that myocardial damage resulted from coronary spasm.

Right coronary arteriography was not performed after each electrical discharge. Consequently, it is impossible to determine the cumulative effect of repeated high energy discharges on the induction of coronary spasm. The presence of post-shock ST segment elevation was not unique and did not differ from findings in other patients receiving intracardiac shocks for ablation of ventricular tachycardia or the AV junction. We have routinely observed this response in a majority of patients receiving high energy intracardiac shocks, but have not performed routine acute coronary angiography in this group of patients.

Although the mild inferior wall hypokinesia viewed angiographically 3 days after the procedure raises a question concerning the significance of induced coronary spasm, it could have resulted directly from the electrical discharges rather than from transient ischemia secondary to epicardial coronary constriction. We can only speculate concerning the pathophysiologic mechanism of ST segment elevation and the frequency of coronary spasm caused by electrode catheter ablation procedures.

Conclusion and recommendations. If ultimately proved effective and safe, electrode catheter ablation could become a significant therapeutic option for the treatment of patients with preexcitation, particularly when accessory pathways are localized to the posterior interatrial septum. However,

this single case raises a serious question regarding the application of the technique to free wall accessory pathways. The potential for procedure-induced acute coronary spasm and possible late complications resulting from the acute coronary artery injury appear real. Because of this experience, we believe that patients undergoing an accessory pathway ablation procedure should have initial baseline coronary arteriography and both early and late post-procedure coronary arteriography to further define the potential for coronary damage. A continuous intravenous infusion of nitroglycerin or the use of intracoronary nitroglycerin during ablation procedures may be warranted. Perhaps attempts at free wall accessory pathway ablation should be limited to those patients whose immediately adjacent coronary artery is nondominant or supplies limited functioning myocardium.

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